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\title{The effects of mindfulness and fantasizing on rumination and MDD: A network perspective}

\shorttitle{Master Thesis - Kaiser}

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\abstract{}

\keywords{Major Depressive Disorder, rumination, network analysis, experience sampling, mindfulness}

\begin{document}

\maketitle

\section{Introduction}

About one in ten people currently suffer from Major Depressive Disorder \parencites[MDD;][]{kessler2013epidemiology, tolentino2018dsm}, and more than one in five will be affected by this debilitating mood disorder at some stage in their lives \parencite{hasin2018epidemiology}. Furthermore, MDD is a recurrent disorder, meaning that individuals that have endured one episode, commonly experience further ones in the future. In fact, the likelihood of recurrence is greater than 80\% after 15 years \parencites[][]{hardeveld2010prevalence, mueller1999recurrence}.

According to the \textit{Diagnostic and Statistical Manual of Mental Disorders} \parencites[5th ed.; DSM-5;][]{american2013diagnostic}, depression encompasses behavioral, emotional and cognitive symptoms (see Table~\ref{tab:MDD}). Cognitive symptoms that are routinely observed in depressed patients such as poor executive control, attentional biases toward negative information, and impaired working memory, are sometimes viewed as mere epiphenomena of the symptoms of the emotional symptoms. However, a meta-analysis by \textcite{rock2014cognitive} shows that cognitive impairments remain detectable even in-between low-mood episodes. Accordingly, cognitive theories highlight the importance of cognitive factors in the onset and maintenance of depression \parencite{kircanski2012cognitive}.

\subsection{Rumination}

A concept that commonly takes a leading role cognitive theories of depression is \textit{rumination}. Rumination is a form of repetitive negative thinking, and as such, is characterized by a repetitive, uncontrolled stream of negatively-valenced thoughts and memories that follow a common theme \parencite{lyubomirsky2015thinking}. In particular, rumination is focused on the past or the present, revolves around themes of loss, meaning, and lack of self-worth, and involves viewing events as certain and uncontrollable \parencite{nolen2008rethinking}. Importantly, rumination links cognition with the behavioral and emotional symptoms of depression. It has been shown to exacerbate and prolong negative affective states such as sadness, anger, and depressed mood \parencites{lavender2004rumination, lyubomirsky1998effects, lyubomirsky1995effects, lyubomirsky1999ruminators, rimes2005effects}. Similarly, it has been demonstrated that both increased negative affect and decreased positive affect predict subsequent rumination \parencites{hjartarson2021daily, moberly2008ruminative}. These bidirectional effects lead to a vicious cycle between affective and cognitive symptoms, with each amplifying the other \parencite{ciesla2007rumination}. In individuals prone to depression, the mutual intensification between negative thinking (including rumination itself) and negative mood produces a forceful downward spiral, eliciting severe levels of negative affect. The vicious cycle between rumination and negative mood is only one pathway by which rumination can lead to and prolong depressive episodes, however. Ruminators are at risk for social isolation, both because they might shun social interactions themselves and because they may be perceived as unwelcome company. Indeed, rumination has been linked to a number of undesirable interpersonal characteristics, such as dependency \parencite{spasojevic2001rumination}, neediness \parencite{spasojevic2001rumination}, and sociotropy \parencites{gorski2002sociotropy, nolen2001mediators}. By eroding social support, rumination increases an individual's risk of slipping into depression. Additionally, rumination interferes with sleep, both lowering sleep quality and increasing sleep latency {--} the time it takes somebody to fall asleep \parencite{thomsen2003rumination}. Rumination, moreover, has been linked to increased anxiety \parencites{kirkegaard2006association, nolen2011heuristic}. However, this relationship may be indirect, with rumination affecting levels of anxiety primarily via its effects on other negative affective symptoms, such as anger and sadness \parencite{thomsen2003rumination}.

Recently, \textcite{watkins2020reflecting} proposed the H-EX-A-GO-N model to explain the onset and perpetuation of rumination as a consequence of the interplay of some or all of five key mechanisms. According to this model, at its core, rumination is an initially functional response to perceived goal discrepancies (GO), but over time can become a mental habit (H) and triggered by low mood. As a result, rumination ultimately becomes dysfunctional, interfering with rather than stimulating efforts to reduce perceived goal discrepancies. The acquisition of mental habits by repeated occasions of rehearsal and conditioning is a central route for the development of pathological rumination \parencite{watkins2014habit}. Crucially, however, the model hypothesizes that this route from occasional, healthy rumination (or state rumination) to excessive, pathological rumination (or trait rumination) is facilitated and made more (or less) likely through the effects of the other mechanisms.

A lack of executive control (EX) facilitates the development of pathological rumination. Individuals with poor executive control may struggle to disengage from repetitive thinking about negative life events and personal concerns. This mechanism is supported by substantial neurobiological evidence. MDD is associated with a weak prefrontal cortex (PFC), so-called "hypofrontality". The PFC's inadequate control results in a hyperactive amygdala \parencites[for example,][]{siegle2007increased,johnstone2007failure}. As a consequence, people suffering from MDD recover more slowly from psychological stress than non-depressed individuals. Neurologically, this manifests itself as more enduring elevated post-stress cortisol levels, which over time can damage the hippocampus \parencite{steffens2011change}. This neurobiological chain reaction not only results in episodic negative changes in mood but also several cognitive deficits, among them executive control \parencite{rock2014cognitive}. Executive control may be viewed as consisting of three main components: shifting, updating, and monitoring representations in working memory and inhibition of irrelevant information \parencite{miyake2000unity}. As \textcite{koster2011understanding} indicate, impairment of such executive functions may contribute to rumination in at least two ways. First, problems with monitoring and manipulating information in working memory might result in the proliferation of irrelevant, negatively-valenced information in working memory, making it evermore salient and harder to disengage from. Such poor cognitive control manifests itself as mental "stickiness" -- uncontrolled and perseverative cognition \parencite{joormann2011sticky}. Second, as executive control is needed to instigate and maintain goal-directed behavior, the weakening of it may result in difficulties in overriding ruminative habits (H) that have formed.

In addition to the effects of poor executive functions, pathological rumination is accompanied by a preferential bias for processing negatively-valenced information \parencite{gotlib2010cognition} as well as an engagement bias that orients attention towards negatively-valenced information and a disengagement bias that hampers the ability to shift attention away from it \parencites{whitmer2013attentional, beckwe2016attentional, donaldson2007rumination}. Such biases, in turn, increase the frequency and accessibility of negative thinking, and therefore, prolong rumination \parencite{watkins2020reflecting}. Stated more generally, rumination and depression appear to be linked to a universal bias for negatively-valenced information \parencite[N;][]{dalgleish1990biases}.

The final mechanism is particularly important to understand why rumination becomes maladaptive. Pathological rumination is characterized by an abstract processing style (A). That is, a processing style focused on generalized and decontextualized mental representations that convey the causes, consequences, and meaning of a goal or an event (the "why" aspects). Unlike the more adaptive and concrete processing style of functional rumination, however, it neglects the feasibility, mechanics, and means of goals and events \parencite[the "how" aspects;][]{watkins2008constructive}. Consequently, pathological rumination fails to alleviate goal discrepancies (GO), leading to longer and more frequent periods of rumination, further promoting habit formation (H).

Given the myriad of associations between rumination and other variables, it is not surprising that rumination's sphere of influence is not restricted to MDD. To the contrary, rumination is associated with an increased risk for social phobia \parencite{mellings2000cognitive}, PTSD \parencite{mayou2002posttraumatic}, and symptoms of generalized anxiety disorder \parencite{watkins2009depressive}. Furthermore, it has been strongly implicated with substance abuse, alcohol abuse, and eating disorders \parencites{aldao2010emotion, caselli2010rumination}. As a result, \textcite{nolen2011heuristic} have proposed a transdiagnostic model featuring rumination as a primary risk factor for multiple disorders. The precise psychopathology that ultimately manifests is determined by specific moderators (e.g., environmental context factors or biological characteristics). Instead of improving our understanding "only" of depression, consequently, rumination may be a cornerstone in an improved conceptualization of a range of mental disorders. However, because it is such a complex construct, tying together affective and cognitive factors, a holistic understanding of rumination requires researchers to adopt a wider lens, investigating not the relationships it forms with individual symptoms, but its role in a complex network of interdependent variables. A more comprehensive and integrated understanding of rumination may have major clinical implications, increasing our ability to predict outcomes such as the course of illness, probability of relapse, and treatment response.

\begin{table\*}[]

\caption{Criteria for an MDD diagnosis. In addition to the \textbf{required criteria}, at least five of the \textbf{depressive symptoms} must be present for at least two weeks. Furthermore, at least one of the symptoms marked with \* has to be observed.}

\label{tab:MDD}

\begin{tabular}{{l}{l}} \toprule

\textbf{Depressive symptoms} & \textbf{Explanation} \\ \midrule

Depressive mood\* & Most of the day; near-daily \\

Loss of interest / pleasure\* & Most of the day; near-daily \\

Weight loss or gain & Change of >5\% body weight in a month\\

Insomnia or hypersomnia & near-daily \\

Psychomotor agitation or retardation & near-daily; observable by others \\

Fatigue & near daily \\

Feelings of worthlessness/guilt & near-daily; guilt may be delusional \\

Lack of concentration & near daily; observable by others or merely subjective \\

Thoughts of death or suicide & not just fear of dying; suicidal ideation without a specific plan \\ \midrule

\multicolumn{2}{l}{\textbf{Required criteria}} \\ \midrule

\multicolumn{2}{l}{Symptoms cause significant distress or impairment in social, occupational, or other important areas of functioning.} \\

\multicolumn{2}{l}{Episode not attributable to physiological effects of a substance or another medical condition.} \\

\multicolumn{2}{l}{Episode not better explained by, for example, schizophrenia, delusional disorder, or other psychotic disorders.} \\

\multicolumn{2}{l}{No history of manic or hypomanic episode.} \\

\bottomrule

\end{tabular}

\end{table\*}

\subsection{Network Analysis}

Network analysis is an analytical framework that allows us to study the complex, interdependent interactions of multiple symptoms as one integrated system. It has entered the field of psychopathology as a result of the perceived shortcomings of the current diagnostic tools such as the DSM-5 \parencite[for a discussion, see][]{borsboom2013network}. Instead of viewing mental disorders as being the result of an underlying root cause (analogous to Western medicine), it conceives of them as a network of interacting elements that do not need to share a root cause. Given that many mental disorders share symptoms, the established boundaries between them might need to be redrawn. Network analysis might lead to greater precision in the delineation of mental disorders and a more realistic understanding of comorbidities in terms of bi-directional symptom-to-symptom connections \parencite{cramer2010comorbidity}. Rumination may turn out to feature prominently not just in a network of depression but in a grander network structure explaining a number of mental disorders conventionally regarded as (entirely) separate entities.

More generally, network analysis is concerned with the measurement, description, and visualization of relational structures \parencite{furht2010handbook}. The end product of such an analysis is typically a graph consisting of both nodes and edges that join them. While each node represents some entity moving within the network, each edge represents a direct connection between two entities. Some connections are likely stronger than others. This is commonly accounted for by varying the thickness and/or color of edges according to {--} for example {--} the number of times two entities are linked with each other. This is referred to as the weight of an edge. Additionally, edges can be distinguished by whether they are directed or undirected, i. e. whether a connection between two nodes always runs from entity A to entity B or can go both ways. In an organization, a directed edge could indicate a well-defined chain-of-command, while in a network of symptoms it would indicate that there is a causal effect from symptom A to symptom B. These connections, then, must be explained by some underlying biological or psychological process. A connection between insomnia and fatigue, for example, can reasonably be explained by some underlying biological process. By comparison, a connection between depressed mood and feelings of worthlessness is likely of a psychological nature.

\subsection{Previous research}

The network perspective on psychopathology resonates with many researchers. As a result, many studies have already used network analysis to study depression. Recently, \textcite{malgaroli2021networks} have systematically reviewed such studies. They attempted to combine the networks created by the 23 reviewed studies into one, specifying the most recurrent centrality and edge weight indices.

Depressed mood and fatigue were the symptoms with the most robust connections across all reviewed studies. Consequently, both of these symptoms appear to be central to MDD. Neither of these findings should be surprising, given that depressed mood is the hallmark symptom of MDD and fatigue has previously been shown to be one of the main protagonists in MDD \parencite{cramer2010comorbidity} and may even be an early sign of its onset \parencite{contreras2019study}. Interestingly, at least one of the two symptoms is necessary to diagnose a depressive episode with ICD-10’s MDD diagnostic algorithm \parencite{ac016969671993icd}, underscoring their pivotal roles in the disorder.

In terms of associations between variables, depressed mood and anhedonia exhibited the strongest connection. This result aligns neatly with the scientific literature, suggesting this link to be a consequence of the dopaminergic pathway and its role in initiating MDD \parencite{stein2008depression}. Feelings of worthlessness exhibit robust connections with other affective symptoms too. \textcite{malgaroli2021networks} consider this to be consistent with MDD’s high comorbidity rates with conditions involving such experiences as guilt, particularly post-traumatic \parencite{lee2001role}, obsessive-compulsive \parencite{shafran1996guilt}, and alcohol/substance use disorders \parencite{treeby2012shame}. Their review, however, was limited in that it only included cross-sectional studies. As such, their network reveals nothing about the causal directions between two symptoms, limiting the ability to make inferences on the dynamics of MDD. It is also noteworthy that the studies reviewed by \textcite{malgaroli2021networks} {--} even the ones they excluded from their review {--} rely on self-reported measures. Of course, many symptoms are not readily measured by objective means. However, cognitive symptoms like lack of concentration would permit objective measurement, potentially increasing a network's accuracy.

Other studies used longitudinal data but with large intervals between repeated measurements \parencite[for example, two years;][]{savelieva2021longitudinal}. Important information about the short-term dynamics of a network is necessarily lost at this time scale. Even (near-)daily measurements \parencites[for example,][]{groen2019capturing,lydon2019adolescent} may be too infrequent to detect some information of clinical relevance. MDD network studies that collected measurements multiple times per day using the Experience Sampling Method (ESM) are rather rare. An exception is the study by \textcite{hoorelbeke2019between} who collected self-reports six times a day for one week from 85 remitted depressed patients. They looked at key vulnerability and protective factors in a network of symptoms with a focus on indicators of emotional (dys-)regulation (mainly rumination and positive appraisal), cognitive functioning, and resilience. One of their main findings was that resilience is an important factor in obtaining stable remission from depression, predicting weaker depressive symptomatology and lower levels of rumination. Like most current network studies, the study by \textcite{hoorelbeke2019between} did not include a healthy control group, limiting their ability to draw conclusions based on their results.

%Furthermore, positive affect acted as the key mechanism by which resilience extended its positive effects.

\subsection{The present study}

The present study builds on and extends previous network analysis studies of depression. Our literature review divulged two gaps that we want to address in the current study. First, even though \textcite{hoorelbeke2019between} included rumination as a node in their network analysis, they assessed it using only two items ("focused on feelings", "focused on problems"). In our opinion, these two items are insufficient to characterize a participant's extent of rumination as they do not reveal the perceived unpleasantness or stickiness of current thoughts, and do not clearly distinguish rumination from other forms of repetitive negative thinking, such as worry. Even in-between depressive episodes remitted patients typically experience residual depressive symptoms \parencite[for example,][]{nierenberg2010residual}. In addition, compared to healthy controls, remitted MDD patients report higher levels of repetitive negative thinking such as rumination \parencite[for example,][]{mcmurrich2008dispositional}. Thus, in an initial step, we create a temporal network of depressive symptoms for remitted MDD patients. To improve upon a common limitation of current network studies, our study includes a control group of healthy individuals. Second, so far no study has investigated the effects of common interventions on rumination from a network perspective. Two interventions that have proven successful at alleviating depressive symptoms are mindfulness and positive fantasizing. While mindfulness focuses on accepting and letting go of negative thoughts, fantasizing focuses more on stimulating positive thoughts. Mindfulness-based therapy has received ample attention and its efficacy in mitigating depressive symptoms has been corroborated by multiple meta-analyses \parencite[see for example,][]{hofmann2010effect}. Positive fantasizing is a main constituent of Preventive Cognitive Therapy {--} a therapeutic approach that has been shown to successfully prevent the recurrence of depressive episodes and mitigate depressive symptoms \parencite{bockting2009long}. It has been suggested that mindfulness exerts its mitigating effects on depressive symptoms by reducing dysfuntional emotion regulation strategies, such as rumination (Guendelman et al., 2017). Returning to XY’s HEXAGON model of rumination, we can identify a likely route this effect might take. Rumination is suggested to be a maladaptive habit (H). A habit can be unlearned. However, this requires conscious effort, i.e., executive control (EX).

Positive fantasizing may, by contrast, exert its effects rather by increasing positive affect (Van Tol et al., 2020 🡪 find better citation; not published?)

%write about how mindfulness may exert its effects by reducing ruminative tendencies --> jury is still out but likely... --> talk about HEXAGON --> specifically H, EX (+GO and N?). Use Hertel, 2004 for H and EX specifically

In fact, both of them may exert much of their effects by intervening in ruminative tendencies.

Our literature review revealed two limitations in current network studies. First, a lack of control groups that limits the conclusions that can be drawn from a network. Consequently, this study includes a and (2) a reliance on self-reported measures.

The current study combines ESM data (collected multiple times a day) and data from a sustained attention to response task \parencite[SART;][; collected once per day]{robertson1997oops}. ESM refers to the collection of self-reports regarding a participant’s ongoing experience \parencite{kahneman2004survey}. Typically, participants are asked to report on their internal experiences and the context in which they occurred at random or set intervals while going about their regular lives \parencite{smallwood2006restless}. While ESM is used to collect self-reports, i.e., subjective measures, SART is a method for obtaining objective cognitive data. Developed by \textcite{robertson1997oops}, SART is a go/no-go paradigm that measures, as the name suggests, the ability to sustain attention. The SART involves pressing a key in response to frequently presented non-targets (Go trials) and withholding the response to infrequently presented targets (No-Go trials). Because it places very low demands on controlled processes, the SART lends itself to mind-wandering, a form of perseverative, repetitive thinking. And indeed, participants' error rates on the task are predicted by questionnaires on mind-wandering \parencite{smallwood2006encoding}.

%Further, response latencies are associated with errors in SART, and the probability of errors occurring is related to blocks of trials in which mind wandering is reported, but not by blocks of trials in which subjects report being on task \parencite{smallwood2004subjective}. In addition to pre-error speeding, there is also post-error slowing which may reflect several task-related cognitive actions, such as re-engagement of the task after the mind has wandered \parencite[for example,][]{rabbitt1966errors}, a return of attention to the external environment \parencite{smallwood2006encoding}, or even negative mood \parencite{smallwood2009shifting}.

%In keeping with the spirit of MINDCOG, the current study combines the subjective and objective measures obtained via ESM and SART and analyses them holistically.

\begin{figure}[]

\centering

\includegraphics[scale=.125]{figures/Hypothesis network.jpg}

\caption{A simplified network of some of the main hypothesized effects. Bigger boxes indicate greater centrality. Red arrows indicate a diminishing influence, green arrows an increasing one. The thickness of the arrow gives an indication of the expected effect size.}

\label{fig:hypothesisNet}

\end{figure}

With this data, this study aims to answer two main research questions. The first concerns the role of rumination in a complex network of symptoms of MDD:

%Although a multitude of studies have investigated the network of symptoms of MDD, most use cross-sectional data only and focus on the criteria defined in the DSM-5, and therefore, neglect rumination and its cognitive symptoms. Thus, the first research question can be stated as follows:

\bigskip

\noindent Q1: What is the role of rumination in a complex network of symptoms of MDD and how does it differ between remitted MDD patients and healthy controls?

\bigskip

The ”role of rumination” is operationalized by typical measures used in network analysis. Its relative importance is measured using centrality measured such as strength. In addition, its influence on other symptoms may be operationalized as the weight of its connections with them. The network properties of the networks for remitted MDD patients and healthy controls can be compared using the Network Comparison Test \parencite[NCT;][]{van2017comparing}.

The second research question concerns the effect of mindfulness and positive fantasizing on the network of symptoms. It may be stated as follows:

\bigskip

\noindent Q2: What are the effects of mindfulness and fantasizing on the network of symptoms of MDD?

\bigskip

To answer this question, a comparison between different measures such as centrality, connection weights, and global strength before and after each intervention as well as network comparison tests will be utilized. In addition to these main research questions, we may also investigate individual resilience and risk factors for episodes of MDD and how self-reported symptoms (ESM data) relate to objective measures of cognition (SART data).

Network analysis, as implemented today, is mainly an exploratory tool \parencite{borsboom2021network}. Strong conclusions should not be drawn on the mere basis of network analysis, but need to be supported by other statistical analyses and substantiated theoretical reasoning \parencite{bringmann2022psychopathological}. For that reason, we designed a tentative "hypothesis network" for remitted MDD patients prior to having received an intervention based on the evidence reviewed in previous sections. Figure~\ref{fig:hypothesisNet} shows the hypothesis network. It was reduced to some of the (likely) most central nodes. Moreover, nodes that may be analyzed separately were placed into categories (e.g., positive affect, negative affect). The network indicates that rumination (or a combination of features akin to rumination) is expected to feature prominently in the network. Negative affect, and to a lesser degree positive affect (or at least some of their constituent parts), are also hypothesized to be highly central to the network. Strong bi-directional effects are expected between rumination and sleep as well as positive and negative affect. Worry is likely to be highly predictive of anxiety.

For healthy individuals we expect negative affect and especially rumination to be less central and many of the associations to be significantly weaker, possibly to the extent that they do not feature in the network at all, resulting in a sparser network (reduced global strength). Similarly, after having gone through an intervention, the networks of remitted MDD individuals might resemble that of healthy individuals more (though they will still be significantly different). Mindfulness may impact the network by reducing rumination directly, whereas fantasizing might exert its influence by increasing positive affect.

\section{Methodology}

Following \textcite{borsboom2021network}, we can regard network analysis as consisting of three main phases: network structure estimation, network description, and network stability analysis (see Figure~\ref{fig:NA\_steps}). The initial phase, network structure estimation, comprises two steps. First, one has to select the nodes to feature in the network. This step is primarily driven by theoretical considerations, rather than methodological ones. Second, using statistical tools, the conditional associations between nodes are estimated to determine the most important edges in the network. Towards this goal, several statistical methods can be employed. Model selection methods based on fit indices, such as regularized estimation procedures \parencite{epskamp2018tutorial}, null hypothesis testing procedures, and cross-validation approaches are commonly used in psychological studies. These approaches typically use conditional associations to define a network structure underlying a set of variables \parencite{robinaugh2020network}. Two variables are said to be conditionally associated if they are probabilistically dependent, conditional on the remaining variables in the set. The strength of this conditional association determines the edge weight between the two variables. If the association between two variables vanishes when the other variables are controlled for, the variables, i.e., their corresponding nodes, will be disconnected in the visualized network structure.

The next phase, network description, involves describing the topology of the estimated network. We differentiate between global and local network topology. Globally, the most important topological feature of a network is the density of its connections. If the number of edges relative to the number of nodes is low, the network is said to be sparse. Conversely, a high number of edges as compared to nodes indicates a dense network. This distinction is critical because some estimation procedures are more suitable for sparse networks, some for dense ones. Another way to think about a network's density is sometimes referred to as global strength. In psychopathology networks, it's a measure of total symptom connectivity, or edge weights, and is hypothesized to be a central factor in an individual's vulnerability profile. \textcite{cramer2016major}, for example, simulated MDD networks, varying the strengths of connections between symptoms. They found that more strongly connected networks are more likely to be tipped into a state of depression as compared to more loosely connected networks. This is in line with findings from other areas of science that show that strongly connected dynamic systems are more easily tipped from one state into another \parencite[e.g.,][]{chen2012detecting} and with the observation that successful therapeutic interventions are sometimes targeted at weakening symptom-to-symptom relations (such as exposure therapy). Global strength can be measured, for example, via the Network Comparison Test \parencite[NFT;][]{van2017comparing}. The most commonly investigated local feature of a network's topology is node centrality. It measures how important a node is. "Importance" within a network can be defined in different ways, and consequently, there are many measures for this property \parencite{das2018study}. The most basic of these is referred to as degree, which measures how many connections a node possesses. In psychological research, strength {--} the sum of all absolute edge weights a node is directly connected to {--} is in high demand for specifying a node’s centrality as it is generally more stable than other common metrics \parencite{bringmann2019centrality}. Other local features of interest include clustering of nodes into subcommunities, providing insight into potentially unobserved causes and the dimensionality of the system, and shortest paths between nodes, possibly yielding insights into the most predictive pathways within the network. Frequently, network analysis involves the estimation of multiple networks, using multiple statistical methods with different advantages, or comparing groups. Hence, the comparison of different networks is another step in the network description phase.

The purpose of the final phase, network stability analysis, is to ensure reproducibility (obtaining the same conclusions from the same data) and increasing replicability (obtaining the same conclusions from new data) of the estimated networks {--} topics that have received ample attention in the psychometric network literature recently \parencite[see for example,][]{bringmann2022psychopathological}. The main targets of statistical tools for robustness analyses in networks are individual edge weight estimates, differences between edges in a network, and topological metrics, such as node centrality. In determining the robustness of edge weight estimates, measures of sensitivity to sampling errors, such as confidence intervals, credibility intervals, and bootstrapped intervals are indicated. The degree to which such intervals overlap for the relevant coefficients gives insights into the robustness of differences between edge weights. To investigate the robustness of other network properties, such as node centrality, various approaches have been devised, including approaches based on bootstrapping \parencite{epskamp2018estimating} and Bayesian statistics \parencite{williams2020bayesian}. The final product generated with these steps is then interpreted. To make substantive inferences, however, it is necessary to combine the output with general methodological considerations and domain-specific knowledge \parencite{borsboom2021network}.

Naturally, the interpretation of a network is dependent on the data used to inform its structure. Generally speaking, we can either use cross-sectional or longitudinal data. In network studies within the fields of psychology and psychopathology, cross-sectional data is most commonly used \parencite{borsboom2021network} {--} resulting in \textit{contemporaneous networks}. Estimating contemporaneous networks is simpler than estimating networks from longitudinal data because for cross-sectional data independence of measurements can typically be assumed. Since independence is given, the application of population-sample logic is warranted, lowering the requirements for an estimation model. In time-series data, two kinds of interdependence are introduced that complicate the estimation of \textit{temporal networks}. First, data collected on two (or more) subsequent measurement points may be correlated (if somebody is happy right now, there is a high chance they are still happy in two hours). Second, responses from one person may correlate with one another more strongly in general (somebody of a more happy nature will on average report more happiness). The obvious downside of contemporaneous networks, however, is that they contain no information about the direction of effects between variables. Additionally, some researchers have voiced concerns regarding the replicability of contemporaneous networks \parencite[for example,][]{fried2017moving}, and have argued that they cannot necessarily be assumed to generalize to the level of the individual \parencite[for example,][]{bringmann2018don}. Overall, temporal networks may be more advantageous than contemporaneous networks. Although the violation of independence assumptions places greater demands on models, the additional insights that can be extracted from it may well be worth the effort. Vector autoregressive models \parencite[see for example,][]{haslbeck2015mgm} have been devised to deal with temporal data. When using such models to estimate a temporal network, one ends up with a structure of associations that remain after taking the temporal effects into account \parencite{borsboom2021network}. Therefore, when using time series data, one receives two network structures, one depicting lagged associations {--} or temporal effects {--} and a kind of contemporaneous network that depicts the associations unaccounted for by the temporal effects. The temporal network, generally, can be interpreted in terms of carry-over effects at the timescale defined by the time between repeated measurements. The temporal ordering may allow for a causal interpretation. It is important to note, however, that causal interpretations are not straightforward \parencite{fried2017moving}. The contemporaneous network will capture effects that occur at timescales different from those defined by the spacing between repeated measurements.

\begin{figure\*}[]

\centering

\includegraphics[scale=.5]{figures/NA\_steps.png}

\caption{Schematic representation of the workflow used in network analysis. Adapted from \textcite{borsboom2021network}.}

\label{fig:NA\_steps}

\end{figure\*}

% ######################################################################

%\section{Methods}

%To achieve these aims, the study utilizes data collected in the wake of a larger project at the University Medical Center Groningen (UMCG) and the University of Groningen (RUG). For each cohort of participants, data was collected for at least eight weeks. Throughout the duration of the project cognitive, behavioral, and (neuro-)physiological measures were gathered from remitted MDD patients and healthy controls. The experiment is split into five periods: two pre-intervention periods, two peri-intervention periods, and one wash-out period. The experiment starts with a pre-intervention period of one week. Thereafter, in the first peri-intervention period, participants receive one of the two interventions at random. This period also lasts one week, with participants receiving a two-hour-long professional training pertaining to their respective interventions on the first day of the period. For the remainder of the period, participants are asked to perform a 10-minute home exercise of the same intervention daily. This is followed by a wash-out period of at least one month. Subsequently, participants go through another round of pre- and peri-intervention periods, this time receiving the intervention they had not yet received.

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\appendix

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%Rumination is a core mechanism of MDD and can explain the development and maintenance of negative affective states. However, rumination is not bound by our definitions of mental disorders. Therefore, to better understand rumination, and by extension depression, we must look at the bigger picture, beyond the margins of MDD as defined by the DSM-5. Instead, we need to investigate it more holistically, taking into account not only affective symptoms (of MDD) but also cognitive ones.

%As we shall see in section (\nameref{section:ruminationNetwork}), substantial evidence supports rumination's link to many undesirable outcomes. However, so far this body of evidence largely relies on studies looking at individual relationships or small sets of relationships.

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